Clinical Evaluation of Artificial Embolization in the Management of Large Cerebral Arteriovenous Malformations*

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Presented here is an interim appraisal of the potential clinical usefulness of artificial embolization in the surgical management of certain large cerebral arteriovenous malformations. In prior communications\(^{4,5,6}\) we have stated a rationale for this procedure, discussed observations of the responses of cerebral arteries to plastic emboli and described a technical modification permitting controlled catheterization of the proximal cerebral arteries. Here we will describe certain anatomical features of the lesions relating to artificial embolization and give a detailed account of our clinical experience to date. This includes observations in 15 patients, from a series of 55, in whom a total of 620 emboli were introduced.

Anatomy of Arteriovenous Malformations

The possibilities for embolization are dependent upon certain anatomical changes associated with these malformations. Since the introduction of angiography, it has been shown repeatedly that to many of the large hemispheric lesions there are enlarged arterial channels leading directly from the cervical arteries. In 1957, Hamby\(^2\) published a gross microscopic dissection of a hemispheric malformation indicating that these enlarged arteries terminate at the malformation by ramifying into multiple smaller interconnecting arteries before passage into a series of tortuous venous channels. This suggested that large emboli within the feeding arteries would arrest at this site which is the vascular origin of the malformation. Because the arteries to the normal surrounding brain remain of normal caliber, or smaller, a large difference in diameter between these and the feeding arteries exists. Therefore emboli sufficiently small to traverse the length of the feeding arteries could at the same time be too large for entrance into these smaller normal arteries.

For purposes of embolization we classify the large hemispheric lesions anatomically within the framework of the normal cerebral arterial anatomy. This is based upon the 55 cases referred to us since initiation of this study. By serial angiography it is usually possible to separate three distinct components of vascular alteration. These are:

1. Primary abnormality of direct artery to vein shunt.
2. Secondary enlargement of the feeding arteries.

Angiography demonstrates that the arteries and veins involved are those that normally supply and drain this vascular zone. Furthermore, the anastomotic relationship of the feeding arteries is similar to that of the normal cerebral collateral circulation, as has been described by Van der Eecken.\(^8\)

Normally, there is a paucity of collateral connections between adjacent cortical branches arising from each of the primary divisions of the internal carotid artery. In arteriovenous malformations this is also true, for small cortical arteries, adjacent to the feeders, pass around the malformation with no apparent participation.
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Multiple leptomeningeal arteries form the normal collateral connections of the anterior, middle and posterior cerebral arteries. When the anterior middle and posterior cerebral arteries jointly contribute to a malformation, they do so through the leptomeningeal arteries which are increased in size but not in number.

Usually there are very limited collateral connections between the penetrating arteries over the surface and the leptomeningeal arteries at the base. In arteriovenous malformations these arteries participate jointly only when the zone of abnormal shunting extends between the territories they normally supply.

The anatomical characteristics of the draining veins have been described by Kaplan et al.\(^8\) The cone-shaped appearance of the larger malformations conforms to the anatomical configuration of the transcerebral veins which normally join the superficial and deep venous systems but in arteriovenous malformations are transformed into much larger venous channels. When a single deep vein assumes the major burden of drainage it may enlarge into an aneurysmal sac (venous aneurysm). This is more frequently seen in the great vein of Galen or veins adjacent thereto.

The prototypes of the classification we use are shown in Fig. 1.

In Type A the lesion occupies the basal ganglia, internal capsule and thalamus and is supplied by multiple, enlarged leptomeningeal arteries. The main trunk arteries bypass the lesion. Malformations of this type cannot be treated by embolization and direct surgical attack is not reasonable.

Type B is supplied by surface arteries and small penetrating arteries. Embolization can eliminate only the surface artery contribution. However, this may be of clinical value in certain instances as suggested by one of our patients whose progressing hemiparesis was arrested for 1½ years following embolization which accomplished only this much.

Malformations of Type C are the most favorable for embolization. However, when the lesions are large there is always some contribution from one or more enlarged leptomeningeal arteries to the medially extending apex thereby precluding complete elimination of the lesion. Also the leptomeningeal anastomotic arteries from the anterior cerebral artery are potential feeders after elimination of the middle cerebral contribution. In our 3 cases with this type of lesion angiography revealed renewed filling of a portion of the lesion via these routes 1 to 3 years after embolization.

In type D, which is equally supplied by anterior and middle cerebral arterial branches, there is a good chance for elimination of the middle cerebral contribution but the anterior cerebral poses a problem. Although it is desirable that the emboli seek the anterior and middle routes equally, our observations indicate a division of about 10 to 1 in favor of the middle cerebral. The reasons for this will be discussed below.

We have not, as yet, attempted to embolize malformations of Type E in which the entire angiographic contribution is from both anterior cerebral arteries. Although these arteries may become fully as large as the middle cerebral trunks, in most of our cases this has not sufficiently altered the angles of branching at the bifurcation of the internal carotid artery to offset the more direct continuation into the middle cerebral trunk.

Occipital malformations conforming to Type F must be embolized via the basilar system. Our single experience with this will be described below.

Of the arteries proximal to the malformation, there are 2 sites of major importance in embolization. These are the internal carotid artery bifurcation and the division of the middle cerebral trunk into 2 or more principal Sylvian arteries. The most frequent alterations in arterial diameters and configurations secondary to malformations occurring at these sites are shown in Figs. 2 and 3.

Most commonly the middle cerebral trunk, in direction and size, is the anatomical continuation of the internal carotid artery; the anterior cerebral artery is half as large and arises at an acute angle. When the
malformation is in the middle cerebral artery territory emboli of appropriate size will remain in the internal carotid middle cerebral artery continuum with absolute certainty. The dominant influence of this directional continuum is further seen in cases with both anterior cerebral and middle cerebral arterial contributions. In our observations emboli easily fitting into either of these branches will choose the straighter course into the middle cerebral artery in about 90 per cent of cases. However, complete reversal of this size differential and directional continuum may occur with very large malformations in the anterior cerebral artery distribution. Also it may occur at the internal carotid artery bifurcation of the side opposite the malformation when there is generous cross contribution. In a single case with this extreme anatomical alteration the emboli pursued the anterior cerebral route uniformly.

Large malformations below or above the Sylvian fissure greatly alter the directional

FIG. 1. The six prototypes of large hemispheric arteriovenous malformations based upon the relationships of the normal, feeding and anastomotic arteries.

FIG. 2. The middle cerebral artery is the direct anatomical continuation of the internal carotid artery and the anterior cerebral artery arises as a branch. With arteriovenous malformations supplied equally by these arteries both enlarge but the general configuration is unaltered. When the anterior cerebral artery is the only feeder, it becomes the continuation of the internal carotid artery and the middle cerebral artery becomes the smaller branch.
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Fig. 3. With malformations above the Sylvian fissure the feeding arteries enlarge and become directly continuous with the middle cerebral trunk. The remaining Sylvian arteries become smaller side branches. With malformations below the Sylvian fissure the reverse occurs.

continuum and size differentials of the middle cerebral branches. The Sylvian branches not participating in the malformation are converted to small side branches, and when the size differential is 2 to 1 or greater, the emboli will almost certainly pursue a course to the malformation.

Surgical Technique

Bilateral carotid and vertebral angiographic studies are performed in all patients considered for embolization. Because of the increased blood flow it was necessary, in 2 cases, to use increased concentration of contrast media (Hypaque 70 per cent) to achieve adequate definition. The side injected is placed directly against the X-ray cassette to minimize magnification. The extent of the lesion and the feeding arteries, including the most proximal trunks, are mapped out with accurate measurements of diameters and assessment of sites of branching or atheromatous narrowing. A correction of 10 per cent is made for image magnification.

Initially, hand-molded spheriods of methylmethacrylate containing a fragment of steel or tantalum were used as emboli. Later molded spheres of marlex plastic were employed and most recently molded silastic (silicone rubber) spheres containing a steel ball. The silastic has the consistency of soft rubber, and multiple lengths of silk suture can be sewn through each sphere to project from the surface about 1 mm. to encourage thrombosis over a wider area in the malformation.†

General anesthesia is now used exclusively. The carotid artery is exposed through a diagonal incision along the anterior border of the sternocleidomastoid muscle. Great care is necessary to prevent sacrifice or compressive occlusion of the adjacent enlarged draining veins. Usually it is possible to introduce a soft, thin-walled plastic catheter of appropriate inner diameter into the lumen of the common carotid artery using the external carotid artery as a sleeve. When this artery is too small, the catheter can be introduced directly into the common carotid artery. This technique is shown in Fig. 4.

Our silastic emboli range in size from 1.0 mm. to 6.0 mm. in increments of 0.5 mm. Generally the smallest size is tried first. This is at least 1 mm. larger than the arteries to normal brain. If the site of arrest is satisfactory, embolization can proceed and the emboli may be introduced singly or in groups of 2 or 3. Polaroid films are taken after each introduction and at appropriate intervals angiography is performed. A critical point is

† Made by Brunswick Manufacturing Co. Inc., 90 Myrtle St., No. Quincy 71, Massachusetts.
reached when the flow to the malformation becomes greatly reduced as the sites of entrance to the feeding arteries are clogged with emboli. The emboli should be introduced only one at a time thereafter, and the procedure terminated if delayed passage of emboli to the malformation occurs. After removal of the plastic catheter the arterotomy is closed with continuous arterial sutures. Usually the wound is drained for 24 hours. X-rays are repeated at frequent intervals during the first days postoperatively to disclose any delayed changes in position. The arteriograms are repeated prior to discharge.

**Clinical Observations**

The disposition of the 55 patients referred to this service since initiation of this study (Table 1), suggests the relative clinical usefulness of embolization. Twelve underwent direct surgery either because the malformation was small or embolization was not feasible. Among these there was a single operative mortality, this being a 3-year-old boy who died of cardiac arrest after apparent successful obliteration of the large supranecephalic malformation that had caused aneurysmal enlargement of the great vein of Galen.

Twelve patients underwent embolization in 1 or more stages as the only surgical procedures. In 2 of the earlier cases the procedure was terminated prematurely, but in all the others an effort was made to obliterate as far as possible all remnants of the malformation. All of these malformations except 1, were hemispheric lesions which had been considered inoperable because of location or size.

A patient in whom it was possible to eliminate all angiographic evidence of the malformation will now be described in detail.

**Case 1.** A 28-year-old white man was transferred to the Mount Alto Veterans Administration Hospital on April 29, 1961, following a spontaneous intracerebral hemorrhage.

Approximately 3 weeks previously a severe right-sided headache had begun abruptly, followed immediately by progressive weakness of the left arm and leg. He did not lose consciousness but the weakness of the left side progressed to a complete flaccid hemiplegia within a few hours. He was admitted to a hospital where a lumbar puncture revealed grossly bloody spinal fluid under increased pressure. A right percutaneous carotid arteriogram demonstrated the probable presence of a very large saccular aneurysm in the course of the right posterior cerebral artery. Also, there was a mass in the right parietal area consistent with an intracerebral hematoma. The lesion was considered inoperable and there-

**TABLE 1**

<table>
<thead>
<tr>
<th>Disposition of patients with arteriovenous malformations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classification</td>
</tr>
<tr>
<td>---------------------------------------------------------</td>
</tr>
<tr>
<td>Direct surgery only</td>
</tr>
<tr>
<td>Embolization only</td>
</tr>
<tr>
<td>Embolization plus direct surgery</td>
</tr>
<tr>
<td>Referred back for surgery or embolization</td>
</tr>
<tr>
<td>All surgical treatment deferred</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>
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Fig. 5. Antero-posterior and right lateral carotid arteriograms showing the arteriovenous malformation in the posterior temporal horn supplied by the choroidal arteries. The draining vein is enlarged into a venous aneurysm. The basilar artery fills via the posterior communicating artery.

after he showed no change in his neurological status to the time of his transfer 3 weeks later.

Examination. Blood pressure was 110/80. The general physical examination was not remarkable. He was normally alert and mentally active. The pertinent neurological findings were a left spastic hemiparesis including the face. There were trace voluntary movements of the toes and ankle but none of the wrist or fingers. There was severe left homonymous hemianopia. He showed astereognosis and dermatognosia in the left hand. There was no right-left confusion. Position sense in the left arm was grossly impaired.

The chest x-ray revealed moderate left ventricular enlargement. Plain x-rays of the skull showed thickening of the calvarium and calcifications in the falx. There was an endosteoma in the posterior fossa and scattered calcifications near the midline on the right side. A crest of this appeared to outline the wall of the large aneurysm. Bilateral carotid and right vertebral angiography were done, Fig. 5. This demonstrated a large venous aneurysm in the right posterior temporal horn supplied by one arterial feeder from the internal carotid artery, probably the anterior choroidal artery, and a branch of the right posterior cerebral artery which joined this internal carotid artery feeder.

Operation. On July 21, 1961, he underwent embolization via the right internal carotid artery. A single 3.0 mm. embolus was introduced and it arrested precisely at the point of origin of the venous aneurysm. Angiography immediately thereafter, Fig. 6, demonstrated absence of the sac.

Fig. 6. An arteriogram immediately after the introduction of one 3.0 mm. embolus which arrested at the terminus of the anterior choroidal artery (indicated by the arrow).
Postoperative course was uneventful and he continued to show gradual improvement in left hemiparesis. Twenty days following surgery angiograms were repeated on the right carotid and right vertebral arteries, Fig. 7. There was normal arterial filling and no evidence of the malformation. He was discharged 35 days following surgery. At that time he was able to walk with forearm crutches and a left leg brace. The left homonymous hemianopsia was unchanged. He convalesced at home with continuing improvement but has never been able to return to work as a clerk. On January 28, 1963, he was readmitted to Mount Alto Veterans Administration Hospital and the right vertebral and carotid angiograms were repeated. Again the arteriovenous malformation did not fill.

In all the remaining cases some portion of the malformations could be demonstrated in postoperative serial angiography. Mostly this persistent filling was via smaller penetrating arteries or the surface leptomeningeal anastomotic channels from the anterior cerebral or the posterior cerebral arteries. In three patients direct surgical approach followed partial embolization. The first of these had a large malformation in the right occipital lobe supplied principally by the posterior cerebral artery but receiving substantial contribution from the surface anatomic channels of the middle and anterior cerebral arteries. Embolization restricted to the internal carotid artery eliminated the middle cerebral contribution only and the lesion was subsequently excised in stages elsewhere. In the 2nd patient with a very large posterior frontal lesion the direct approach was used to occlude some of the feeding branches from the anterior cerebral artery after almost total elimination of the middle cerebral arterial contribution by embolization. The 3rd case was that of a 12-year-old boy with a large malformation of the dura and dural sinuses. He will be described in detail (Case 4).

Seventeen cases were sent back to the referring neurosurgeons with recommendations for direct surgical excision or embolization. The 11 remaining cases were considered untreatable. These were mostly large malformations deep in the hemisphere or brain stem and principally supplied by numerous penetrating arteries (Type A). The large number of cases (20 per cent) considered
TABLE 2
Symptomatic follow up after artificial embolization

<table>
<thead>
<tr>
<th>Principal Symptom</th>
<th>No. of Patients</th>
<th>Time since embolization (yrs.)</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemorrhage</td>
<td>5</td>
<td>1, 2, 4, 4, 5 1/2</td>
<td>No recurrences</td>
</tr>
<tr>
<td>Headache</td>
<td>3</td>
<td>2, 4, 6 (1 not followed)</td>
<td>Continuing alleviation</td>
</tr>
<tr>
<td>Progressive hemiparesis</td>
<td>1</td>
<td>3</td>
<td>No further progression</td>
</tr>
</tbody>
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untreatable is not, however, representative of arteriovenous malformations in general because many of the cases referred were selected from other centers after a direct surgical approach had been rejected.

Eight patients have been followed for an interval greater than 1 year following embolization, Table 2. In 5 with a history of 1 or more cerebral hemorrhages there have been no recurrences during intervals ranging from 1 to 5 1/2 years. In all except one of these it was possible to demonstrate some refilling of the malformation by follow-up angiography at intervals of approximately 1 to 2 years. The only patient presenting a progressive hemiparesis has shown no further progression after a period of 2 1/2 years. Of the three patients with intractible head pain, two have been followed for 2 and 4 years with significant continuing improvement.

Complications of Embolization

The complications encountered have generally fallen into the six categories in Table 3. Most of these have been remedied by minor alterations in technique and increasing experience.

Thrombosis at the tip of the plastic catheter used for irrigating the emboli into the internal carotid circulation occurred in 3 occasions when the catheter had been introduced directly into the lumen of the common carotid artery. The thrombosis progressively ensnared the emboli preventing intracranial progression. In all 3 it was necessary to terminate the procedure and retrieve the emboli and clot. However, in each instance the procedure was resumed at a second stage and there were no untoward consequences. The use of the external carotid artery as a sleeve for the catheter has eliminated this complication.

Spastic narrowing of the internal carotid artery immediately proximal to the carotid canal in the petrous bone occurred in 1 patient near the end of a long procedure. It was necessary to terminate the procedure but the intended result was obtained.

Three patients with very large malformations had saccular aneurysms arising from one of the major feeding arteries. Two of these, whose aneurysms arose from the internal carotid artery were treated, without complication, by permanent occlusion of the cervical portion of the internal carotid artery after completing the embolization. The 3rd patient with a great vein of Galen malformation had a large arterial aneurysm near the terminus of the basilar artery. This ruptured fatally, 2 hours following the last of 5 stages of embolization via both internal carotids and basilar systems. It had not been recognized preoperatively, having been considered a venous dilatation adjacent to the great vein of Galen. The rupture apparently resulted from a change in intravascular pressure after the fistulae had been occluded.

Passage of an embolus into an artery not leading to the malformation occurred with 1 embolus in 1 case. Prior to embolization several attempts had been made elsewhere to operate upon the large malformation in the left frontal region. Though the major feeding arteries had been clipped, the malformation continued to fill through many smaller arteries. The differential in size between these and normal arteries was less than 2 to 1. The aberrant embolus passed into one of the normal Sylvian arteries and lodged in the inferior parietal lobe of the dominant hemisphere. On the 3rd day after

TABLE 3
Complications of artificial embolization

1. Thrombosis at tip of irrigating catheter
2. Spasm of internal carotid artery
3. Rupture of associated saccular aneurysm
4. Passage of emboli into normal artery
5. Passage of emboli through malformation
6. Proximal arrest of emboli
operation the patient complained of transient numbness of his right hand but this was not substantiated objectively, and there was no clinical evidence that significant infarction had occurred.

Passage of emboli directly through the malformation with ultimate arrest in the venous sinuses or pulmonary circulation occurred in 4 patients. The first of these was a patient with a large midline malformation (the patient with the great vein of Galen malformation and basilar aneurysm already described above). There were direct fistulous connections to the great vein of Galen from both posterior cerebrols, the terminal anterior cerebrols and the basilar artery. The patient was embolized in 5 stages via both internal carotid and vertebral arteries. Because there was no small vessel phase between the arteries and veins, embol as large as 4.0 mm. passed through the fistulae immediately or after several hours delay. Ultimately, the fistulae were nearly completely occluded but 53 emboli ranging from 3.0 to 4.0 mm. passed into the lung fields. No significant cardiopulmonary sequelae were noted. The absence of a small vessel phase between feeding arteries and draining veins in certain midline malformations, particularly those associated with aneurysmal enlargement of the great vein of Galen, has been established by careful pathological dissection. Accordingly, this complication had been anticipated in this patient but in the other 3 patients with large mid-hemispheric lesions the progression of emboli completely through the malformations had not been anticipated. The emboli passing through in these cases ranged from 2 to 6 in number and from 3.0 to 3.5 mm. in diameter. The sites of passage were restricted to only 1 feeding artery and emboli in the other feeders arrested at the origin of the malformation. It was ultimately possible to eliminate these sites of direct shunting by employing larger emboli. No cardiopulmonary sequelae occurred. In all of these patients one or more emboli arrested at various sites in the draining venous sinuses. No explanation is given for this unusual finding. There was no evidence that this was associated with partial obstruction of the sinuses.

Transient or permanent arrest of emboli in feeding arteries proximal to the malformation was the most important cause of complications. It occurred in 6 patients near the conclusion of the procedure. In 3 no deficit was produced. In 2 a transient deficit occurred but in the last it led to a mortality directly attributable to embolization. This patient, with a very large occipital malformation primarily supplied by the right posterior cerebral artery, presented a long history of intractible headaches and a progressive left homonymous hemianopia. He was embolized via the right vertebral artery. After 45 emboli measuring 2.5 and 3.5 mm. in diameter had passed into the malformation an arteriogram showed some persistence of filling from more proximal branches of the posterior cerebral artery. Five more emboli were introduced one at a time and repeat x-rays showed that the last 4 had arrested at the terminus of the basilar artery. The 1st of these 5 had completed the occlusion of the remaining contribution from the posterior cerebral artery. The diminished blood flow resulting therefrom was insufficient to carry the remaining 4 into the distally obstructed artery. Recurrence of this complication can be prevented, we believe, by more judicious introduction of emboli near the end of a procedure.

The following detailed report describes a patient with a suitable situation for embolization but in whom many of the above complications were encountered.

Case 2. A 55-year-old white male caretaker was referred to the Clinical Research Unit at Georgetown University Hospital on November 25, 1963, with a diagnosis of inoperable arteriovenous malformation of the left parietal lobe.

Approximately 20 years prior to admission he experienced the first of a series of transient episodes during which he was unable to articulate. These episodes lasted from 10 to 15 minutes and were occasionally associated with conjugate deviation of the eyes to the right and repetitive muttering of simple words. He never lost con-

* Referred by Drs. G. Stoll and W. Cotter, Providence, R. I.
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sciousness. Over the ensuing years the episodes increased in frequency finally occurring every 2 to 3 months. About 4 months prior to admission he experienced an episode which, for the first time, was associated with a severe left-sided headache and nuchal rigidity. These symptoms cleared in about 48 hours but 2 months later a similar episode prompted admission to a hospital. A lumbar puncture demonstrated bloody spinal fluid. A left percutaneous carotid arteriogram showed a large arteriovenous malformation in the left parietal area. It was considered inoperable and he was discharged on dilantin, 100 mg. 4 times a day. A week before admission he awoke in the morning with weakness and numbness of the right arm and leg and difficulty in articulating such that he could utter only short, simple phrases. A repeat lumbar puncture failed to demonstrate recurrent bleeding and he was transferred to the Clinical Research Unit at Georgetown University Hospital for further work-up.

Examination. Blood pressure was 110/70. The patient was right handed, normally alert, cooperative and in no distress. In his ordinary conversation he showed moderately severe paraphasia. He could follow simple commands and showed no right and left confusion. There was no perseveration. In reading he mispronounced approximately every other word and he could not read difficult words at all. There was moderate impairment of simple calculating. There was no anemia. A continuous bruit with systolic accentuation was audible over the left mastoid, eye and neck. He had a right incongruous homonymous hemianopia with central sparing. There was a slight right lower facial weakness. Skilled motor acts of the fingers of both hands were normal. The deep tendon reflexes were equal bilaterally. There were no sensory or motor disturbances in the extremities. His gait was normal and there were no abnormal cerebellar signs.

Routine laboratory tests were within normal limits. X-rays of the chest showed normal cardiac size. X-rays of the skull showed hyperostosis frontalis and occipitalis and slight sellar enlargement. An electroencephalogram showed a moderate to marked abnormality with rhythmic slowing and spikes maximal in the left temporal region and flat polyomorphic delta activity in the left parieto-occipital and posterior temporal regions. Bilateral carotid and right vertebral angiography was performed. The left carotid arteriogram demonstrated a large arteriovenous malformation supplied by multiple enlarged Sylvian arteries and a single enlarged lenticulostriate artery (Figs. 8–11). The right carotid arteriogram showed faint filling of a superior medial portion of the malformation via enlarged leptomeningeal anatomic branches. Angiographically, the basilar system contributed to the malformation via a greatly enlarged posterior communicating artery, but there was no contribution from the terminal branches of the posterior cerebral artery. On the left carotid arteriogram there was filling of normal arteries to the posterior frontal lobe and these measured approximately 0.8 mm. in maximum diameter. The enlarged Sylvian arteries feeding the malformation ranged between 3.5 and 5 mm. in average diameter.

On December 5, 1963, the first stage of embolization was performed under general anesthesia. Singly or in groups of 2, 4 emboli measuring 3 mm. and 9 emboli measuring 4 mm. were introduced. Twelve of these lodged at various sites at the origin of the malformation. The 13th embolus, measuring 4 mm., passed through the malformation into the lungs, and the procedure was terminated. Postoperatively, his clinical status was unaltered. The bruit was still audible. X-rays on successive days demonstrated no change in position of the emboli and confirmed the single embolus in the pulmonary circulation. On December 10, 1963, embolization was repeated. A single 3.5 mm. embolus was introduced. This also passed through the malformation. Then a five zero silk suture was attached to a 4.0 mm. embolus and the embolus introduced with sufficient length of the suture to permit ascent only to the origin of the malformation. At this site the embolus was held stationary. Ten 3.5 mm. emboli were introduced one at a time. These arrested in the malformation, 7 of them immediately proximal to the 4.0 mm. embolus which was occluding the site of direct artery to vein communication. However, about 3 minutes thereafter, the 4.0 mm. embolus shifted position slightly and the 7 emboli passed through into the adjacent draining veins. The attached silk suture was severed and permitted to flow into the carotid circulation, and the procedure terminated.

Postoperative Course. Over the ensuing 24 hours 6 of these 7 emboli progressed distally, 3 into the lungs and 3 into the right sigmoid sinus. There was no change in the patient’s neurological status. However, on the 2nd day after operation he had abrupt recurrence of headache and the appearance of anoma, finger agnosia, acauleia and alexia. There was slight awkwardness in skilled movements of the right fingers. A spinal tap revealed a pressure of 220 mm. of cerebrospinal fluid, which was moderately bloody, indicating another small hemorrhage from the malformation. Over the ensuing 10 days there was gradual improvement and he was discharged.

Readmission. While convalescing at home his improvement continued and he was readmitted on March 18, 1964. Examination showed moderately severe paraphasia, slight anoma and moderate impairment in simple calculations and reading.
Skilled movements of the right hand were still slightly impaired. The bruit was audible but slightly reduced from his previous examination. Visual field examination continued to show a right incongruous homonymous hemianopia with central sparing. A left carotid arteriogram showed essentially the same degree of filling of the malformation. On March 25, 1965, he underwent a third stage of embolization. A total of 18 emboli measuring 4 mm. were introduced and none of these passed through the malformation. However, the last 3 arrested proximally as near obliteration of the malformation was reached and one of the previously introduced 3.5 mm. emboli worked through into the Sylvian draining veins. Angiography showed some persistent filling through the enlarged lenticulostriate artery (Figs. 12 and 13), which could not be occluded by embolization. The procedure was terminated.

Postoperatively there was no change in his clinical status. The bruit was no longer audible over the left eye. His postoperative convalescence thereafter was uneventful. The left carotid arteriogram was repeated on April 6, 1964, and showed no appreciable change from his immediate postoperative films. Neurological examination at the time of discharge showed no change.

The patient has been followed for 5 months. There have been no recurrent episodes of speech arrest or hemorrhage, but he has had 2 generalized seizures.

For malformations in the temporal lobe it may be desirable to block the feeding arteries at their origin from the middle cerebral trunk. In this instance arrest of the emboli proximal to the lesion should be intentional. A case confirming this will be described in detail.

Case 3. A 45-year-old white man was admitted to the Neurosurgical Service at Georgetown University Hospital on May 16, 1964 with a diagnosis of a cerebral arteriovenous malformation.*

He had been in good health until 3½ months prior to admission when he experienced the rapid onset of a severe headache and left hemiplegia followed by loss of consciousness. He was admitted to a hospital where angiograms demonstrated a large arteriovenous malformation occupying the right temporal lobe and an intracerebral clot in the parietal area. A craniotomy was performed and the clot removed. Postoperatively there was gradual improvement and he was discharged ambulatory 26 days following surgery. Thereafter he was aware of a continuous bruit and his wife noted frequent brief periods when he seemed out of contact.

Examination. Blood pressure was 139/80. The patient was right handed; general physical examination was not remarkable. He was fully alert, but occasionally confused as to time and place. He had a moderately severe nominal dysphasia. A continuous bruit was easily audible over the right eye, forehead and temple. The right external jugular vein was very prominent and the right carotid pulsation was more pronounced than the

* Referred by Drs. W. Cotter and G. Stoll, Providence, R.I.
Fig. 10. This phase shows the nidus and draining veins. The drainage is to the sagittal sinus and the Sylvian venous system.

left. He had a complete left homonymous hemianopia. He showed a slight left-sided spastic hemiparesis most marked in the forearm and the hand. There was impairment of skilled movements of the fingers of the left hand. He showed right-left disorientation, impaired two-point discrimination and markedly impaired proprioception in the left arm and hand. He exhibited extinction of simultaneous stimulation everywhere over the left side and tended to fall to the left

Fig. 11. An antero-posterior view of the left carotid arteriogram. The arrow indicates a single enlarged lenticulostriate artery supplying the apex of the malformation.

Fig. 12. A lateral view of the left carotid arteriogram immediately after the final stage of embolization. The arrow indicates the lenticulostriate feeder which was not occluded. Three emboli have arrested proximal to the malformation.

Fig. 13. An antero-posterior view of figure 16 showing persistent filling via the enlarged lenticulostriate artery (arrow). Three emboli in the right sigmoid sinus and a single embolus in a Sylvian vein on the left can be seen.
with Rhomberg testing.

**Laboratory Studies.** Blood and urine studies were normal. The fasting blood sugar was 100 mg. per cent and the blood urea nitrogen 13 mg. per cent. An electroencephalogram showed a moderate right hemispheric abnormality with widespread depression of voltage and local slowing in the temporal region. X-rays of the chest were normal.

Bilateral carotid and vertebral serial angiograms were performed. The right carotid arteriogram (Fig. 14), demonstrated an arteriovenous malformation occupying most of the right temporal lobe. It was not possible to delineate precisely the configurations and size differentials of all of the branches of the middle cerebral trunk, but the films suggested that the nidus was supplied by the temporal polar, the anterior temporal and posterior temporal arteries. Also there was a contribution to the medial extending apex of the lesion via several enlarged lenticulostrate arteries. A Sylvian artery conforming to the ascending trunk could not be demonstrated. Venous drainage was into the Sylvian veins, cortical veins to the sagittal sinus and via the anastomotic vein of Labbé to the lateral sinus. There was no drainage into the deep venous system. The left carotid angiogram showed some filling of the lesion via the anterior communicating artery to the right middle cerebral trunk and the basilar system contributed via the posterior communicating artery and right middle cerebral trunk. Considering the normal Sylvian arteries to be approximately 1.5 to 2.0 mm. in diameter (not proven angiographically), there was a satisfactory size differential between these and the feeders which ranged from 3.0 to 3.5 mm.

**Operation.** On May 28, 1964, the patient underwent embolization via the right internal carotid artery. Seven emboli measuring 2.5 mm. were introduced singly. Three of these passed through the posterior temporal artery occluding its contribution to the posterior portion of the nidus. Two arrested near the nidus and 2 others passed through the malformation arresting in a draining vein adjacent to the sagittal sinus. Then 3 emboli measuring 3.0 mm. were introduced singly. The 1st of these arrested in a middle segment of the posterior temporal artery, the 2nd immediately distal to the middle cerebral artery division, possibly temporal polar artery, and the 3rd at the origin of the posterior temporal artery. The arteriogram immediately thereafter (Fig. 15), showed satisfactory elimination of the malformation with preservation of the ascending Sylvian trunk which filled with contrast media for the first time. A portion of the malformation continued to fill via an enlarged lenticulostrate artery. The procedure was terminated.

**Postoperatively** the patient showed no change in neurological status and the bruit was no longer audible. However, over the ensuing week it gradually reappeared and on June 5, 1964, the right carotid arteriogram was repeated. It was
Artificial Emboli for Arteriovenous Malformations

Fig. 15. Emboli block the major cortical feeding arteries but not the circulation through the ascending Sylvian trunk (arrow). The malformation fills via an enlarged penetrating artery (arrow).

Fig. 16. Arteriogram seven days following embolization showing distal shift of an embolus (arrow) and complete filling of the malformation.

Fig. 17. The embolus indicated by the arrow again obstructs the origin of the major feeding artery.

noted that the 3rd 3.0 mm. embolus had shifted more distally in the posterior temporal artery and again the malformation filled almost as extensively as before embolization (Fig. 16). The patient was discharged on June 6, 1964.

Convalescing at home he showed gradual improvement. Nearly normal strength returned to the left side, but he continued to show right-left disorientation. He was never able to bathe, dress or shave himself and had periods of severe depression.

Readmission. On September 7, 1964, he was readmitted to the Clinical Study Unit at George-town University Hospital. Examination revealed persistence of the complete left homonymous hemianopia. A continuous bruit was again easily audible over the right eye and temple. The left hemiparesis was improved with only slight impairment of skilled movements of the fingers. Graphesthesia and stereognosis were normal in the left hand and there was no extinction to simultaneous right and left stimulation. However, there was slight impairment of two-point discrimination in the left hand. Skilled co-ordinated movements of both hands were moderately impaired. He was unable to button his shirt or tie his tie.

The right carotid angiogram was repeated with findings similar to the postoperative films (Fig. 16). On September 29, 1964, the patient underwent a second stage of embolization. Scarring prevented separation of the internal and external carotid arteries therefore the plastic catheter was introduced into the common carotid artery directly, and aligned to encourage passage of the emboli into the internal carotid artery only. A 3.0 mm. embolus was introduced and arrested immediately distal to the terminus of the middle cerebral trunk, presumably in the posterior temporal artery, Fig. 17. An angiogram demonstrated marked reduction of contrast filling of the malformation. Two subsequent 3.0 mm. emboli passed unintentionally into the external circulation. After readjustment of the catheter two more 3.0 mm. emboli were introduced. These arrested beyond the middle cerebral trunk in the feeding arteries. The angiogram immediately
thereafter showed cessation of filling of the malformation from the surface arteries, but persistent deep filling from an enlarged lenticulostriate artery, Fig. 18. Circulation through the ascending Sylvian branch supplying the cortex above the Sylvian fissure was preserved. The procedure was terminated.

Postoperatively the patient showed no change in neurological status. The bruit was no longer audible but there was a systolic murmur transmitted from the carotid artery secondary to stenosis at the site of repair of the arteriotomy.

The angiograms were repeated 10 days following embolization. It was possible to fill the malformation as before via the lenticulostriate feeder.

A final case is included to demonstrate the use of embolization to block a feeding artery at a specific point proximal to the lesion.

Case 4. A 12-year-old white boy was admitted to Georgetown University Hospital on December 3, 1950, following the abrupt onset of unconsciousness.

He had been well until approximately 4 hours before admission when, while playing with his schoolmates, he abruptly fell to the ground and remained unresponsive. His past history was significant in that he had had frequent episodes of epistaxis since the age of 2 years and a tendency for his neck to bulge anteriorly when he cried. His maternal grandfather had had a large vascular abnormality of the frontal area of the scalp which had gradually increased in size during the last 20 years of his life. He died at the age of 83 years with no evidence of intracranial involvement.

Examination. Blood pressure was 110/80, pulse 90, temperature normal. He was stuporous, occasionally restless and did not respond to voice but briskly responded to pain. A continuous bruit with systolic accentuation was easily heard over both carotid arteries, the occiput and the back of his neck and head. His eyes tended to deviate conjugately to the left. There was early papilledema and slight paresis of the left arm. X-rays of the skull showed generalized thickening of the cranial vault and a striking increase in the grooves of the middle meningeal arteries as they proceeded towards the occiput.

Complete angiography demonstrated an arteriovenous malformation involving the posterior half of the dura of the middle and posterior fossae (Fig. 19). The internal carotid arteries participated via their tentorial branches. The remainder of the supply was from the external carotid arterial systems and the vertebral arteries via their meningeal branches. The straight sinus was markedly dilated as were both lateral sinuses.

Operation. On the day of admission, the right and left external carotid arteries were ligated and divided. Four plastic emboli had been introduced into the left external carotid prior to division. These lodged in the enlarged meningeal feeding arteries on the left. A tracheotomy was performed.

The patient's clinical status remained unchanged and on the day following admission the left thyrocervical trunk and ascending cervical arteries were ligated. Thereafter his clinical status gradually improved and within 3 days he was normally responsive. At this time he showed rapid horizontal nystagmus on left lateral gaze. The paresis of the left arm cleared.

2nd Operation. Eleven days following admission a ventriculogram was performed using frontal burr holes. This demonstrated enlargement of the lateral and third ventricles but the pressure was normal. Two weeks later he underwent bilateral subtemporal craniectomies and ligation and division of the dilated meningeal arteries on each side. One week later all ascending branches from the left subclavian artery and the right vertebral artery were ligated. He was discharged 6 weeks after admission. At that time his neurological examination revealed nystagmus on left lateral gaze and no other abnormality.

Readmission. During his convalescence at home there were no new symptoms or neurological signs. He was readmitted in July, 1960, for a direct attack on the malformation via the posterior fossa. In the first stage a generous posterior fossa craniectomy was performed with removal...
of the lamina of C1. The second stage was planned with profound hypothermia using extracorporeal circulation. When the body temperature had been reduced to 23°C, it was felt that the perfusion rate was not adequate to support him beyond cardiac arrest. Therefore the lesion was approached at that temperature. The dura in the posterior fossa was divided bilaterally including multiple enlarged arteries extending up from the base to the area of the confluens and the lateral sinuses.

The postoperative course was uneventful and he was discharged 20 days following surgery. He was able to return to school and led a relatively normal life. His neurological examination continued to show nystagmus on left lateral gaze. He continued to have frequent epistaxis requiring hospital admission and packing of the nasopharynx on several occasions.

3rd Operation. On November 8, 1964, after he had successfully completed high school, he had a generalized seizure followed by a right hemiparesis and aphasia. He was readmitted to the hospital where a lumbar puncture demonstrated an initial pressure of 260 mm cerebrospinal fluid. The cerebrospinal fluid was crystal clear. Bilateral carotid and left vertebral angiograms were repeated. The tentorial branches of the internal carotid arteries on both sides had increased in diameter and drained directly into the straight sinus. The left vertebral angiogram continued to show substantial filling of the malformation via meningeal branches, and this appeared to be the greatest contribution. Because of the direct continuation of the left vertebral artery into the basilar artery embolization via this route to occlude the multiple feeding branches presented the risk of passage of an embolus into the basilar artery itself. Therefore, it was decided to occlude the left vertebral precisely at the point where it passed through the dura and gave off its major dural arteries. Emboli introduced thereafter would be blocked from beyond this point and would occlude the multiple anastomotic muscular branches more proximal. After careful measurements a single 6.0 mm. embolus was introduced. This occluded the vertebral artery precisely at the desired site. Thereafter 75 emboli measuring 3.5, 4.0 and 4.5 mm. were introduced. These occluded the vertebral artery and its enlarged branches proximally and angiography demonstrated elimination of all fistulous contributions via this route (Fig. 20).

Over the ensuing 7 days he made a gradual complete recovery and during the following 3 months underwent right and left occipital craniotomies as the first stages in an attempt to divide the dural attachments with the feeding arteries to the posterior sagittal sinus, straight sinus, confluens sinuum and lateral sinuses.
Discussion

An important part of this work was to assess whether angiographic information relating to arterial diameters and configurations, and direction of blood flow permits an accurate prediction of the behavior of the emboli of any given size. Certain allowances must be made for the obvious limitations of angiographic information. Uneven magnification prevents very accurate measurement of arterial diameters. Sites of atherosomatous narrowing may be obscured by high concentrations of contract medium. Arterial configurations at sites of branching are not certain with films in only two planes, and these may be obscured by contrast medium in the lesion or by enlarged veins filling in the same phase. Finally, with considerable shunting of blood to the malformation there may be limited or absence of filling of the normal arteries precluding measurement of their diameters. In this latter difficulty it may be necessary to assume average normal diameters. These are approximately 3.0 mm. for the middle cerebral trunk and 1.6 mm. for the anterior cerebral and Sylvian arteries.

To compensate for these limitations a margin for error of about 1.0 mm. was used; i.e. emboli 1.0 mm. larger than any normal artery and 1.0 mm. smaller than the feeding arteries. With adherence to this our experiences indicate that there can be absolute selective accuracy in the choice between normal and feeding arteries but not between individual feeding arteries. The course of emboli through the latter is more related to angles of branching. When the posterior communicating artery is enlarged to approximately the diameter of the internal carotid artery, with the angle of origin still close to 90°, the emboli tend to remain in the straighter course to the middle cerebral artery. At the internal carotid bifurcation with both anterior cerebral and middle cerebral arteries enlarged and preservation of the normal configuration, the distribution of the emboli is roughly 10 to 1 in favor of the middle cerebral artery. At the middle cerebral artery division into 2 or more large feeding
arteries the emboli select courses almost at random.

Our observations confirm the anatomical fact that most of the large feeding arteries ramify into smaller branches right at the beginning of the malformation. However, at various sites in some of the lesions there is a transition of the arteries into draining veins without significant narrowing, and in certain malformations, particularly those associated with marked segmental dilatation of draining veins, all the connections between arteries and veins may be of large diameter.

The normal anastomotic arteries on the cortical surface surrounding the malformation must be particularly well developed. This is suggested by the infrequency of significant infarction when a feeding artery is occluded by an embolus arresting far proximal to the malformation.

Complete angiographic elimination of most malformations will not be possible until the technique has been perfected. However, the anatomical and physiological principles are valid and the technique can be safe in selected patients. It is not possible to predict that partial or near total elimination of a lesion will result in permanent or temporary clinical gain. Current evaluation of any form of treatment is limited by incomplete accurate data relating to the natural course of the disease process. Past attempts to assemble a group of cases with statistical significance have been inadequate because the lesions have not been classified according to salient anatomical and physiological characteristics.7

Summary
The anatomical and physiological circulatory changes associated with cerebral arteriovenous malformations are described. A classification of large hemispheric malformations based upon normal vascular anatomy is presented. Observations made during embolization of 15 patients with large arteriovenous malformation are described. The complications as well as the apparent reduction or elimination of the malformations are discussed.

References